REVIEW

Management of acute ventilatory failure

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Acute ventilatory failure is a challenging yet increasingly common medical emergency reflecting the growing burden of respiratory disease. It is not a diagnosis in itself but the end result of a diversity of disease processes culminating in arterial hypoxaemia and hypercapnia. This review focuses on key management issues including giving appropriate oxygen therapy, treatment of the underlying aetiology as well as any precipitant factors and provision of assisted ventilation if required. Ventilatory assistance can be provided both invasively and non-invasively and the indications for either or both forms of assisted ventilation are discussed. Further emphasis is needed regarding advanced directives of care and clinicians should be aware of ethical issues regarding assisted ventilation.

Respiratory diseases constitute a significant burden of emergency hospital admissions and are responsible for one in four deaths in the UK.[1 2] Up to 20% of patients admitted to hospital with an exacerbation of chronic obstructive pulmonary disease (COPD) present with a respiratory acidosis.[3] Acute ventilatory failure (AVF) is a challenging yet commonly encountered medical emergency requiring prompt diagnosis and treatment to reduce morbidity and mortality. This review describes the management of AVF focusing on the variety of aetiologies presenting in this way, the importance of appropriate oxygen therapy, and ventilatory support including the impact of non-invasive ventilation on clinical practice.

DEFINITION

Ventilatory failure is defined as the inability of the respiratory system to function effectively as a pump normally able to oxygenate arterial blood and eliminate carbon dioxide from the body. This results in arterial hypoxaemia (Pa_{O_2}<8.0 kPa/60 mm Hg) and hypercapnia (Pa_{CO_2}>6.7 kPa/50 mm Hg) and is classified as type 2 respiratory failure. This should be distinguished from a picture of type 1 respiratory failure characterised by the presence of a Pa_{O_2} less than 8.0 kPa and a normal or low Pa $_{CO_2}$ level.

The hallmark of acute decompensated ventilatory failure is the development of respiratory acidosis with an arterial pH<7.35 with coexisting hypercapnia. The presence of significant respiratory acidosis together with tissue hypoxia can induce myocardial arrhythmias and worsen the already compromised function of the respiratory muscles further leading to a life threatening downward spiral.

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AETIOLOGY

AVF is not a disease in itself but the end result of a diversity of processes and failure to appreciate this may lead to suboptimal management. For instance, worsening hypercapnia in a patient with acute severe asthma heralds a potentially catastrophic situation requiring urgent assessment for assisted ventilation. This contrasts with severe COPD where hypercapnia may be seen during the stable state and decompensation is characterised by respiratory acidosis. In addition, conditions such as acute severe asthma or pneumothorax initially giving rise to a picture of type 1 respiratory failure (Pa_{CO_2}<8 kPa with normocapnia/hypocapnia) at presentation may subsequently be complicated by hypercapnia if untreated as a result of worsening VQ mismatching and increasing load on the respiratory muscles eventually resulting in clinical deterioration.

There are a variety of causes resulting in AVF (see table 1). Perhaps the most common presentation seen in daily clinical practice is that of an intercurrent insult complicating a condition characterised by chronic ventilatory failure such as COPD (acute on chronic ventilatory failure). Such insults include infection, pneumothorax, and thromboembolism and these place a greater load on an already stretched respiratory pump resulting in an increased work of breathing, acute decompensated ventilatory failure, and respiratory acidosis. The epidemic of morbid obesity in the Western world is resulting in the increasingly frequent presentation of AVF secondary to obesity-hypoventilation syndrome that is often associated with sleep apnoea.[4–6] The clinician should be wary of the wide range of acute and chronic neurological/neuromuscular diseases presenting with AVF, for example, Guillain-Barre syndrome, motor neurone disease, myasthenia gravis, muscular dystrophies, paraneoplastic syndromes, and other rare myopathies. Chest wall conditions such as kyphoscoliosis, post-polio syndrome, and thoracoplasty as well as spinal cord injuries may all present in acute decompensated ventilatory failure.[7–9] In addition to peripheral causes, the role of central neurological lesions as well as drugs such as opioids and sedatives in any case of acute respiratory depression and ventilatory failure must not be forgotten. AVF may also arise as a result of intracranial trauma (central respiratory

Abbreviations: AVF, acute ventilatory failure; COPD, chronic obstructive pulmonary disease; IPPV, invasive positive pressure ventilation; NIV, non-invasive ventilation; COAP, continuous positive airway pressure; NIPPV, non-invasive positive pressure ventilation; IPAP, inspiratory positive airways pressure; EPAP, expiratory positive airways pressure

depression) as well as chest wall trauma as seen in ''flail segments''. It must also be remembered that acute cardiogenic pulmonary oedema may present as an acute presentation of ventilatory failure. Rarely, obstructive airway lesions, metabolic derangement as a result of thyroid disease, and electrolyte disturbances such as hypophosphataemia may be a contributory or causal factor and the presence of these must always be sought in any unexplained case of ventilatory failure.[10–12]

CLINICAL EVALUATION IN AVF

A detailed history and careful examination in cases may be useful in elucidating an underlying aetiology of AVF as well as determining any precipitating factors. Obtaining an accurate history from the patient may not be possible in all cases of AVF as a result of clinical instability, drowsiness, cognitive impairment, or dyspnoea and in some cases patients may be unaware of their diagnosis. Although symptoms such as breathlessness, cough, and orthopnea may predominate in COPD and cardiogenic pulmonary oedema, they are often absent in AVF secondary to central hypoventilation chest wall and neurological diseases. Unexplained ventilatory failure in a previously fit adult raises the possibility of head injury or drug overdose—either unintentional or as a consequence of illegal drugs. Information from relatives and carers as well as obtaining old case notes or clinic letters is valuable not only in guiding specific treatment but also in determining prognosis and setting a ceiling of treatment.

Signs of hypercapnia include drowsiness, a bounding pulse attributable to peripheral vasodilatation, coma, flapping tremor, and rarely papilloedema. The presence of central cyanosis is suggestive of significant hypoxaemia. Chest wall deformities, a ''flail segment'', gross obesity, and abnormalities on neurological examination such as bulbar palsy, areflexia, muscle wasting; fasciculation, and weakness may be obvious. Pinpoint pupils can be suggestive of opioid toxicity or brainstem disease. Respiratory examination may yield evidence of hyperinflation suggestive of airflow obstruction such as reduced cricoid distance, tracheal tug, indrawing of the costal margins during inspiration. Auscultation of the chest may also be useful, for example, globally reduced air entry in airflow obstruction, unilaterally reduced air entry in pneumothorax, bronchial breathing suggestive of pneumonia with showers of fine, and coarse crackles in fibrosing alveolitis and pulmonary oedema respectively.

The absence of characteristic symptoms and physical signs in a patient at presentation does not exclude AVF and further investigation is needed in all cases.[13] In an acute setting, pulse oximetry has a role in identifying significant hypoxaemia and the presence of an oxygen saturation of less than 92%–93% should prompt further investigation in terms of obtaining arterial blood gas pressures. The use of pulse oximetry in primary care settings as a screening tool for identifying cases of AVF needs further study. However, pulse oximetry data obtained while breathing oxygen should be viewed with caution as they may be falsely reassuring because they do not identify deteriorating acid-base status. Arterial blood gas measurements must be performed in all suspected cases of AVF.

The most important variable to consider when interpreting arterial blood gas pressures during AVF is the pH value or the observed trend in pH rather than the absolute values of Pa_{CO_2} and Pa_{O_2}. It is worthwhile remembering that an acute increase in Pa_{CO_2} of 0.13 kPa results in a decrease of arterial pH of 0.01. Raised arterial bicarbonate (HCO_3) levels suggest a degree of metabolic compensation that may take several days to achieve. The presence or development of respiratory acidosis in cases of acute on chronic ventilatory failure is a potentially life threatening occurrence, necessitating urgent clinical assessment, review of oxygen, and other medical treatment as well as consideration for assisted ventilation.

A chest radiograph may sometimes suggest an underlying diagnosis such as hyperinflation in COPD, pulmonary oedema, etc, or a precipitating factor, for example, pneumonia, pneumothorax, or neoplasm but may not be diagnostic or of adequate quality in an acute setting. The use of peak flow monitoring to detect the onset of respiratory failure in acute neurological presentations such as Guillain-Barre syndrome is unreliable and spirometry measuring forced vital capacity should be used.[14]

Table 1 Commonly encountered causes of acute ventilatory failure in clinical practice

Aetiology of acute ventilatory failure	Common examples
Central respiratory depression	Drugs, for example, opioids, sedatives
	Cerebrovascular disease
	Space occupying lesions
	Central congenital hypoventilation syndrome (Ondine's curse)
	Uncontrolled oxygen therapy
	Trauma
Airway conditions	COPD
	Bronchiectasis and cystic fibrosis
	Acute severe asthma/chronic asthma
	Obstructing airway lesion, for example, tumour
Disorders of extrapulmonary restriction, the thoracic skeleton, and pleura	Obesity-hypoventilation syndrome
	Kyphoscoliosis
	Thoracoplasty
	Flail segment
	Significant pleural effusion
	Pneumothorax (particularly in secondary cases)
Metabolic and electrolyte derangement	Hypophosphataemia
	Thyroid disease
Severe parenchymal lung disease	Pulmonary fibrosis
	Pneumonia
	Acute cardiogenic pulmonary oedema
Neuromuscular disease and conditions involving the respiratory musculature	Acute polyneuropathy, for example, Guillain-Barre syndrome, botulism
	''High'' cervical spinal cord disease
	Polymyositis and other myopathies
	Motor neurone disease
	Muscular dystrophies, for example, Duchenne
	Myasthenia gravis
	Sequelae of poliomyelitis

Box 1 Key principles in the management of acute ventilatory failure

- Identification of the underlying aetiology
- Performing blood gases to determine arterial pH in addition to Pa_{O_2} and Pa_{CO_2}
- Treatment of any precipitant factors
- Optimisation of oxygen therapy (specifying dose, method of delivery, and adequate monitoring of arterial blood gas pressures)
- Appropriate medical management reflecting the underlying aetiology, for example, bronchodilators, corticosteroids
- Consideration for ventilatory support (non-invasive ventilation/invasive positive pressure ventilation) and determining the ''ceiling of treatment''

Box 2 Important practical issues for clinicians to consider and address when managing acute ventilatory failure

- It is not a diagnosis, think of the underlying cause and any precipitant factors (need for detailed history from patient, relatives, ambulance staff, and thorough general physical examination looking for evidence of multisystem disease, for example, neurological signs, trauma, etc)
- The cornerstone lies in prescription and delivery of appropriate oxygen therapy and monitoring of patients including pulse oximetry and arterial blood gases particularly after starting changes in oxygen therapy
- The pH of the arterial blood gas gives us important prognostic information; if respiratory acidosis persists despite controlled oxygen therapy and medical management of the underlying cause consider the need for assisted ventilation
- Non-invasive ventilation reduces the need for intubation as well as improving short and long term survival in cases of respiratory acidosis complicating acute COPD. It is most effective when started early.

When establishing a patient on non-invasive ventilation think:

- I*s this form of ventilation appropriate for the patient* (is it contraindicated?) and what should I do if it fails—that is, *is the patient a suitable candidate for invasive ventilation?* A clear plan should be recorded setting a so called ''ceiling of treatment'' after discussion with senior medical staff, patient, and carers.
- *Where should I be giving non-invasive ventilation*...ICU, respiratory failure unit, general ward, etc? This will depend on the severity of the illness (including degree of acidosis), the underlying cause of acute ventilatory failure as well as the ''ceiling of treatment'' set.
- When considering suitability of a patient for invasive ventilation, do not just consider lung function (''FEV_1'') or make a decision solely on a presumptive diagnosis of chronic lung disease

Box 1 outlines the key principles and box 2 examines important practical issues in the management of acute ventilatory failure.

OXYGEN THERAPY IN AVF

Appropriate oxygen therapy is the cornerstone in the management in AVF. Oxygen, like any other drug, must be correctly prescribed by medical staff. Concentration, mode of delivery, and duration of use should be clearly stated. Although significant degrees of hypoxaemia are potentially dangerous if untreated, the harmful effects of giving uncontrolled ''high flow'' oxygen therapy in AVF leading to respiratory depression, worsening hypercapnia, and respiratory acidosis have been well reported.[3 15] The mechanisms behind this are multifactorial including suppressing hypoxic drive resulting in a reduction in ventilation, increased V/Q mismatching and respiratory ''dead space''.[16 17] This occurs in an already compromised system where breathing is rapid and shallow (resulting in decreased tidal volumes and overall ventilation) as a result of worsening dynamic hyperinflation, bronchospasm, infection, and airway oedema. The use of uncontrolled oxygen in AVF commonly begins in the ambulance and often continues during initial hospital assessment and admission. In a study of 918 patients admitted with acute exacerbations of COPD over a 12 month period, of the 155 patients with hypercapnia, 81 had a Pa_{O_2} greater than 10 kPa and a third of such ''hyperoxic'' acidotic patients normalised their pH after reduction of the oxygen concentration.[3] In an audit of 97 patients admitted with an acute exacerbation of COPD, the inhospital mortality was greater with those patients prescribed oxygen concentrations greater than 28% compared with those prescribed concentrations equal or less than 28% (14% *v* 2%).[15] In a significant number of cases of respiratory acidosis, uncontrolled oxygen therapy was given by ambulance staff with potentially harmful consequences during long journeys. In those patients with a history of chronic ventilatory failure such as COPD or obesity-hypoventilation syndrome, it is safer to maintain an oxygen saturation of 90%–92% and arterial pH>7.35 with Pa_{O_2} of >6.5 Pa. This will produce very adequate tissue oxygenation provided the cardiac output is preserved and the haemoglobin is reasonably normal. In such patients controlled oxygen therapy is best given with a Venturi system mask rather than nasal prongs. The importance of regular monitoring after the start or after a change of oxygen therapy in AVF cannot be overemphasised and this entails both monitoring via pulse oximetry and titration of oxygen therapy according to arterial blood gas values.

This approach differs from the management of trauma or conditions such as acute severe asthma and pneumonia where a simple face mask and reservoir bag is recommended with a minimum flow rate of 5l/minutes because of rebreathing of carbon dioxide if air is not adequately flushed from the mask.[18] The concentration of inspired oxygen delivered by nasal cannulas varies considerably with the pattern of breathing and minute ventilation and their use cannot be recommended in the acute setting.[19]

MEDICAL MANAGEMENT IN AVF

Medical therapy in AVF is directed at the underlying condition as well as any precipitating factors. During acute exacerbations of COPD, this entails giving bronchodilators for symptomatic relief by reducing dynamic hyperinflation and bronchospasm.[21] The use of oral corticosteroids in COPD exacerbations accelerates improvement in lung function, shortens hospital stay, and possibly prolongs the time until the next exacerbation.[20] Antibiotics are indicated if there is an associated change in sputum volume or purulence.[21] However, the use of intravenous aminophylline has not been shown to confer significant benefit and when considering the potential for toxicity, therefore use cannot be recommended.[22 23] Respiratory stimulants such as doxapram in acute COPD have been shown to be inferior to non-invasive ventilation in terms of improving gas exchange in the presence of respiratory acidosis complicating COPD with the potential for adverse effects such as agitation.[24] Doxapram may be useful in situations as a ''bridge'' to NIV or where assisted ventilation is refused, deemed inappropriate, or is unavailable.

A common cause or precipitant of AVF is acute cardiogenic pulmonary oedema. The use of diuretics as sole first line management in acute cardiogenic pulmonary oedema has been questioned by several studies highlighting the benefits of intravenous nitrate therapy in the acute setting.[25 26] Where sedatives are thought to have contributed to AVF, appropriate antidotes should be given if necessary, for example, naloxone for opioid toxicity.

ROLE OF ASSISTED VENTILATION

Respiratory acidosis that persists or develops despite optimal oxygenation and standard medical therapy signals the need to consider ventilatory assistance. Assisted ventilation can be delivered both invasively (invasive positive pressure ventilation or IPPV) as well as non-invasively (non-invasive ventilation or NIV). The use of both NIV and IPPV should be regarded as complementary modes of treatment rather than being mutually exclusive. The clinician should be aware of both the benefits and potential limitations of using either approach in clinical practice. One useful strategy is to optimise medical management before atarting ventilatory support, deciding early on the appropriateness of any form of ventilatory support for a particular patient as well as carefully monitoring patients while receiving NIV to promptly identify any deterioration.

Use of non-invasive ventilation in AVF

The concept of providing non-invasive respiratory support in cases of AVF is not new. In 1838, Dalziel described ''an apparatus to promote artificial respiration'' in a case of drowning consisting of a box surrounding the body.[27] The use of negative pressure ''tank'' ventilation or the ''iron lung'' was pioneered by Drinker in 1928 and such devices were extensively used in the management of respiratory failure secondary to poliomyelitis.[28 29] This was superseded by the improvements in the area of IPPV and endotracheal intubation in the 1950s. Although negative pressure ventilation has had resurgence in interest during recent years[30]; NIV is nowadays commonly delivered by the use of positive pressure. This commonly entails the use of continuous positive airway pressure (CPAP; where a constant pressure is set during inspiration and expiration) or non-invasive positive pressure ventilation (NIPPV; where a higher pressure is set during inspiration than expiration). In NIPPV, this inspiratory pressure is termed IPAP (inspiratory positive airways pressure) and the lower expiratory pressure set is termed EPAP (expiratory positive airways pressure). The use of CPAP in an acute setting is most commonly seen in a high dependency or intensive care environment in the management of conditions resulting in type 1 respiratory failure. This contrasts with that of NIPPV, which is most often used acutely in cases of decompensated type 2 respiratory failure.

NIPPV is used most commonly in the acute setting to treat ventilatory failure secondary to COPD and cardiogenic pulmonary oedema; being usually applied by means of a tight fitting nasal or face mask attached to a ventilator with the patient fully conscious. Studies have shown successful application of NIPPV in various locations such as accident and emergency, intensive care units, high dependency units as well as general respiratory wards with attention to staffing numbers and training.[31–33]

The physiological benefits of NIPPV in COPD are seen in terms of reduced work of breathing with the applied positive pressure.[34] This offsets the increased elastic and resistive loads placed on the respiratory muscles created by worsening dynamic hyperinflation and airways disease permitting time for standard medical therapy such as bronchodilators, antibiotics, and corticosteroids, etc, to have an effect. Absolute contraindications to the use of NIPPV include facial trauma or vomiting and relative ones include confusion, impaired consciousness, excessive secretions, and where the risk of vomiting is significant.[35] However, a recent study showed that NIPPV may be used successfully in cases of coma induced by hypercapnia and such cases should be considered on an individual basis in intensive care.[36] Common adverse effects of NIPPV include erythema, soreness and ulceration of the nasal bridge as a result of mask pressure, claustrophobia as well as excessive leakage.[37] The effects of positive pressure in a conscious patient may cause ocular irritation, dry mouth, and gastric insufflation. Dryness of the mouth and throat experienced by patients during NIPPV may be alleviated by using humidification devices. Cases of barotrauma resulting in pneumothorax are uncommon but nevertheless the presence of an undrained pneumothorax is a relative contraindication for NIPPV.[35 38]

An important message from the literature is that the greatest benefit from NIPPV occurs when it is started early in the course of decompensated ventilatory failure.[33 39–41] Clinicians should always formulate a plan as soon as possible after the start of NIPPV regarding the appropriateness of ''stepping up'' to intubation and IPPV in event of treatment failure (setting a so called ''ceiling of treatment''). Ideally such discussions should always involve senior medical staff, the patient, and carers. Practically, it is also important to distinguish ''treatment failure of NIPPV'' reflected by a lack of improvement in arterial blood gas pressure from those cases where patients do not tolerate NIPPV, for example, because of claustrophobia or mask fit.

NIPPV during acute exacerbations of COPD

NIPPV significant reduces the rate of endotracheal intubation, the duration of hospital stay as well as short and long term mortality during exacerbations of COPD complicated by acidotic hypercapnic ventilatory failure.[33 39–41] In a multicentred randomised control trial of 236 patients on general respiratory wards, NIPPV reduced the need for intubation (15% *v* 27%; $p<0.02$) compared with standard care.[33] In addition, inhospital mortality was halved in the NIPPV group (10% *v* 20%; $p = 0.05$). In that study, the benefits of NIPPV became less apparent below an arterial pH of 7.30 and the study concluded that more severely acidotic patients should be treated in an intensive care setting. It has also been reported that the use of NIPPV reduces longer term (one year) mortality and morbidity after an admission with acidotic ventilatory failure.[42] In a meta-analysis of eight randomised controlled studies, NIPPV decreased the need for intubation (relative risk 0.42), fewer complications (relative risk 0.32) decreased likelihood of treatment failure (relative risk 0.51), and reduction in mortality (relative risk 0.41).[43] IPPV carries a significant risk of acquiring nosocomial pneumonia (1% a day) that is not seen with NIPPV and this is the most probable reason for the decrease in complications and mortality.

NIPPV should thus be considered during an exacerbation of COPD complicated by acidotic hypercapnic respiratory failure ($pH<7.35$).[35] The degree of respiratory acidosis determines the likelihood of success with NIPPV as does the place where NIPPV is given.[33] In cases where the pH ranges from 7.30 to 7.35, 80% of cases will improve without the need for NIPPV using standard medical management such as controlled oxygen, bronchodilators, corticosteroids, and antibiotics. However, arterial blood gas measurements must always be repeated after one hour or so and the persistence of a respiratory acidosis after this time should warrant NIPPV. Under such circumstances it has been estimated that 10 patients must be treated to prevent one intubation. In situations where the arterial pH is between 7.25 and 7.30, the use of NIPPV is recommended in addition to standard medical therapy though such patients are best managed in a high dependency setting as up to 50% may not improve. The use of NIPPV has also been shown to lead to a reduction in intubation rates and mortality in patients presenting with a more severe acidosis ($pH<7.25$) although this should be performed in an intensive care setting where intubation and IPPV can rapidly be started in event of treatment failure.[44] It is recognised that the step up to intensive care admission and IPPV may not always be

appropriate in every patient and in such cases a trial of NIPPV has been shown in various studies to reduce inhospital mortality and palliate dyspnoea.[45 46]

Despite the benefits, up to 50% may fail to improve with NIPPV.[47] This may occur in the first few hours after the start (early failure) or up to several days later (so called ''late failures'').[48] Prompt identification of such cases is essential to step up therapy to IPPV if appropriate and arterial blood gas pressures should be checked one and four hours after the start of treatment. A fall in respiratory rate, decreased $Paco_2$, an improvement in pH as well as breathlessness after one and four hours shows a more favourable outcome.[49 50] The presence of a more severe baseline acidosis, greater baseline physiological and neurological impairment, radiological evidence of consolidation, significant comorbidity, and lower body weight were associated with a higher chance of treatment failure.[49] The incidence of ''late failure'' (>48 hours) after successful start of NIPPV has been estimated to be about 20% and this has been associated with impaired functional ability to perform daily activities, metabolic derangement such as hyperglycaemia as well as lower baseline pH.[48]

Use of non-invasive ventilation in cardiogenic pulmonary oedema

Sufficient evidence now exists to recommend the use of non-invasive methods of ventilatory support in the management of AVF complicating cardiogenic pulmonary oedema (CPE) although the optimal mode of ventilation (CPAP compared with NIPPV) has not yet been clearly determined.[51–55] Such modes of ventilation should be an adjunct alongside medical therapy such as intravenous nitrates and diuretics. The use of CPAP led to a reduction in the intubation rate with a trend towards decreased mortality compared with standard medical therapy in several randomised controlled trials. A review of these trials concluded that four patients would need to be treated to prevent one intubation.[56] One trial comparing CPAP with NIPPV in CPE alone was stopped because of an excess rate of myocardial infarction in the NIPPV group.[55] However, on examination of the data, there were a greater number of patients with chest pain in the NIPPV group compared with the CPAP group and a subsequent trial did not show an excess of adverse events.[55 57] Furthermore, the NIPPV group showed greater improvements in arterial pH and $Paco_2$ before the trial was stopped. On a practical note, patients receiving NIPPV for CPE should be closely monitored by trained staff in a higher dependency setting. IPPV should be started if there is not an early improvement in the clinical condition and parameters of gas exchange.

Other applications of non-invasive ventilation in acute AVF

NIPPV is a useful first line management in decompensated acute on chronic ventilatory failure complicating obesity-hypoventilation syndrome, motor neurone disease, myopathies, muscular dystrophies as well as chest wall problems such as kyphoscoliosis and thoracoplasty.[4–9 58] Long term NIPPV improves daytime symptoms, quality of life, and survival in ventilatory failure complicating such conditions.[59–61] The use of NIPPV in the treatment of acute respiratory failure complicating myasthenia fravis or Guillain-Barre syndrome is limited to small intensive care unit cases series, which suggest that NIPPV may prevent intubation under such circumstances.[62 63]

NIPPV should be used with caution (in an intensive environment only) during the management of AVF complicating community acquired pneumonia (CAP). There is a higher failure rates (>50%) associated with NIPPV in CAP and that the benefits of NIPPV here seem to apply mainly to those patients with coexisting COPD.[64] The use of NIPPV in an intensive care unit reduces intubation and mortality in immunocompromised patients with fever, pulmonary infiltrates, and respiratory failure.[65] This may be particularly beneficial as intubation and IPPV carries a significant mortality in this group of patients.[66] NIPPV may also be beneficial in acute severe asthma and AVF secondary to trauma but again, this should be given in an intensive care setting only.[67 68] There is insufficient evidence at present to recommend the widespread use of NIPPV in AVF complicating cystic fibrosis or bronchiectasis where the retention of secretions may be problematic.[35 69 70]

Establishing an acute NIPPV service

Improvements have been seen in the availability of an acute NIPPV services in UK hospital trusts. In 1997, NIPPV was available in less than half of acute hospitals in the UK.[71] However, a telephone survey of 228 hospitals performed in 2003 showed that 96% could deliver NIPPV acutely and that this was available ''out of hours'' in all such departments.[72] Although guidelines on the use of NIPPV have been published, successfully providing an acute NIPPV service entails emphasis on appointing suitable clinical leads, establishing local policy on use, adequate training of junior doctors, physiotherapists, and nursing staff. Such training needs to include greater education regarding appropriate patient selection (selecting those who may receive NIPPV on a respiratory ward from those who need assisted ventilation in intensive care) thus acting as ''gate keepers'' on the front line. This entails a multidisciplinary approach between various departments, for example, respiratory medicine, accident and emergency, intensive care, etc, in addition to focusing on the invaluable part played by specialist nurses and physiotherapists.

Invasive ventilation in AVF

A number of studies have challenged traditionally held concepts regarding the prognosis of chronic lung disease patients undergoing invasive ventilation (such as difficulty in weaning and having a universally poor outcome). In a study of 166 COPD patients undergoing IPPV, the inhospital mortality was 28% and the mean duration of IPPV was 8.9 days.[73] However, this was before the introduction of NIPPV to the centre involved in the study. Inhospital mortality was higher in the presence of comorbidity, malignancy, duration of IPPV>72 hours, and greater derangements in acute physiological parameters but was not related to FEV_1. Mortality was decreased in those who had previously undergone IPPV thus inferring a selection benefit. Only 9% of patients underwent IPPV for>21 days. In a retrospective review of 63 COPD patients (mean FEV_1 0.74l) undergoing IPPV, the median period of ventilation was two days and only 13% received IPPV>one week.[74] Inhospital and one year mortality were 20% and 48% respectively. Here the inhospital mortality was greater in those with a higher $Paco_2$ and again a greater acute physiological derangement but not FEV_1. In another prospective multicentre study of 362 intensive care unit admissions with COPD, the inhospital mortality was 24% and the strongest predictor of which was the presence of non-respiratory organ dysfunction.[75] This and other studies found that the use of IPPV did not predict either short or long term mortality.[75 76]

The decision about whether to offer a patient with chronic lung disease intensive care admission and invasive ventilation should be carefully considered by senior medical staff taking a number of factors into account. This should include information on quality of life, performance status, confirmation of an underlying diagnosis, patient wishes, and advanced directives. Such decisions should not be made solely on the basis of ''the FEV_1'' or a presumptive diagnosis

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of chronic lung disease. The potential benefits should be weighed against the risks of intensive care admission and IPPV, which include endotracheal tube associated pneumonia (1% a day risk), peripheral neuropathy because of immobilisation, various psychiatric sequelae, and the potential for long term cognitive dysfunction and dependence in survivors and these should be explained frankly to patients and carers where possible.

ETHICAL ISSUES REGARDING USE AND WITHDRAWAL OF ASSISTED VENTILATION

Despite the evidence supporting the use of assisted ventilation in episodes of acute ventilatory failure, a recent UK audit showed that 62% of COPD admissions presenting with a respiratory acidosis did not receive ventilatory support in any form.[77] Pessimism still exists about the prognosis of chronic lung disease patients who require invasive ventilation. Such pessimism is reflected in a recent study that showed a pronounced variation between intensivists in their estimates of survival in patients with chronic lung disease and deciding whether to admit such patients to intensive care.[78] Clearly greater consistency is needed by clinicians in the decision making process.

The withdrawal of mechanical ventilation represents another difficult area in patients presenting acutely with ventilatory failure. In one multicentre study of 851 patients receiving IPPV (215 of these for underlying respiratory disease), the physician perception of patient's preferences regarding life support as well as perceiving a poor outcome (<10% survival or future cognitive dysfunction) were strong predictors of withdrawal of assisted ventilation along with the use of pressor agents.[79]

Often treatment options regarding assisted ventilation are discussed only during disease exacerbations. Such discussion seldom gives time for patients, clinicians, and relatives to carefully consider all options in a balanced light. Awareness and use of advanced directives of care among those with chronic lung disease remains low in the UK. However, one study of COPD patients in the USA showed that in the last six months of life, decisions not to receive mechanical ventilation and cardiopulmonary resuscitation increased from 12% to 31% and 40% to 77% respectively.[80] Education programmes are increasingly being adopted in an attempt to improve self awareness of the condition, compliance with treatment, and the degree of patient involvement in the decision making process. These should also focus on issues regarding terminal care, wishes regarding ventilatory support, cardiopulmonary resuscitation, and advanced directives. Ideally, such decisions in those with severe chronic lung disease should be made during the ''stable'' state perhaps in an outpatient environment with carers and relatives also being present. This may entail a variety of disciplines such as intensive care, respiratory, and palliative medicine with specialist nurses playing an important part alongside doctors.

SUMMARY

AVF is a common medical emergency reflecting an ever-increasing burden of respiratory disease in clinical practice. The causes are many and adequate clinical assessment may give clues to the underlying aetiology. Central to the investigation is arterial blood gas analysis with the pH determining treatment and providing important prognostic information. The treatment entails a multidisciplinary approach focusing on optimal oxygen therapy, medical management of the underlying cause, treating any precipitant factors, and utilisation of assisted ventilation. The use of non-invasive ventilatory support has made a significant impact in the management of this life threatening situation. However, important challenges still remain including selection of appropriate candidates for NIV, improving the consistency of decision making regarding intensive care admission, and assessing the impact of ''stable state'' treatment directives.

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FILLER

What's in a word?

Words are symbolic and usually represent realities elsewhere. Doctors make diagnoses ''identifying the nature of an illness'' using words that, if impressive, can be a substitute for thought, understanding, and insight. Some examples.

Doctors use the word ''effusion'' when they should use the word ''liquid''. Granted, most liquids will be effusions but a so called effusion might be pus, a possibility incorrectly excluded from the differential diagnosis by use of the word effusion. And the management is different.

Dermatologists use posh words. Erythema nodosum sounds like a definitive diagnosis but translated into plain English becomes ''red lumps''. Erythema multiforme becomes a ''multiformed red rash'', Pustular dermatosis becomes ''pussy spots'', and Lichen simplex chronicus becomes ''simple persisting tree moss-like appearances.'' Diagnostic definitivity recedes into infinity.

The words ''cerebrovascular accident'' (CVA) are often (wrongly) used to label sudden onset neurological symptoms or signs thereby assuming an accidental cerebrovascular causation. If a CVA is an act of God, then surely it cannot be an accident? CVA often is used when there is no evidence of a cerbrovascular aetiology. If a patient has a CVA this should mean a lesion that manifests on the opposite side of the body—yet ''left sided CVA'' is often (wrongly) used to refer to a left hemiparesis.

Infectious disease physicians, of whom I am one, are no better. We use the label gastroenteritis without any proof that there is inflammation of the gaster or the intestines. But gastroenteritis sounds more impressive than ''vomiting and diarrhoea of presumed infective aetiology.''

I get irritated by referrals that conclude with the words ''Could this be chronic fatigue syndrome?'' as if it were a diagnosis. It is not, it is a syndrome, a label that tells us nothing about the cause and cure. ''But there are diagnostic criteria'' I hear you say. Well, yes, groups have got together to develop research criteria so that patients entered into studies will have similar complaints. The Fukuda criteria for chronic fatigue syndrome comprise four or more of a selection of complaints and the Holmes criteria comprise two major criteria, and for minor criteria six or more of 11 symptoms and two or more of three physical criteria or eight or more of the 11 symptom criteria (have you ever wondered why two and six? Why not three major and 5 of 11 minor criteria?) What criteria were used to formulate these criteria and who determines the selective threshold? Such research criteria should rarely be used for clinical diagnosis because research criteria often select the more severe end of the symptomatic spectrum. Certainly patients who do not fulfil research chronic fatigue criteria may still have chronic fatigue syndrome—whatever that is.

Some doctors retain insight. As a locum GP I asked the senior partner to see a strange rash. ''Pityriasis'' he declared and the patient departed satisfied. I later challenged him on the grounds that the rash was not any pityriasis that I had been taught. He, like most GPs, was a down to earth mortal (hospital doctors are often high flyers with their feet not on the ground) and responded ''I had no idea what the rash was. It will disappear within a few days. No one will be any the wiser. Calling it pityriasis hides my ignorance and reassures the patient.'' Now he was a person in control of his words.

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